

10575712\_STN



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(FILE 'HOME' ENTERED AT 17:37:25 ON 20 DEC 2007)

FILE 'MEDLINE' ENTERED AT 17:37:36 ON 20 DEC 2007

FILE 'MEDLINE' ENTERED AT 18:02:24 ON 20 DEC 2007

L1	100858	S	STROKE
L2	108644	S	MEMORY
L3	2559837	S	1 AND 2
L4	2397179	S	1 (P) 2
L5	892	S	L1 (P) L2
L6	58619	S	DEMENTIA
L7	20677	S	BIOMARKER
L8	256	S	L6 AND L7
L9	231	S	L6 (P) L7

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FILE 'MEDLINE' ENTERED AT 00:42:52 ON 21 DEC 2007

L1	58619	S	DEMENTIA
L2	160020	S	DIFFICULT
L3	989	S	L1 (P) L2
L4	1276369	S	RAT
L5	10	S	L3 AND L4

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(FILE 'HOME' ENTERED AT 15:31:47 ON 20 DEC 2007)

FILE 'MEDLINE' ENTERED AT 15:32:15 ON 20 DEC 2007

L1	58604	S	DEMENTIA
L2	148	S	ALZHEIMERS
L3	58242	S	ALZHEIMER
L4	58242	S	L2 OR L3
L5	1320716	S	GENERAL REVIEW/DT
L6	12040	S	L5 AND L4
L7	10203	S	STATE OF THE ART
L8	40	S	L6 AND L7
L9	68	S	L4 AND L7
L10	0	S	L8 NOT L9
L11	28	S	L9 NOT L8
L12	12040	S	L5 AND L4
L13	9781	S	UNPREDICT?
L14	12	S	L13 AND L12
L15	2169219	S	TREATMENT
L16	3143	S	L15 AND L12

=> log hold

COST IN U.S. DOLLARS

SINCE FILE

ENTRY

TOTAL

SESSION

FULL ESTIMATED COST

14.92

15.13

SESSION WILL BE HELD FOR 120 MINUTES

STN INTERNATIONAL SESSION SUSPENDED AT 15:48:58 ON 20 DEC 2007

1

L16 ANSWER 89 OF 3143 MEDLINE on STN  
 AN 2007442275 MEDLINE  
 DN PubMed ID: 17646621  
 TI Rationale for transdermal drug administration in **Alzheimer**  
 disease.  
 AU Oertel Wolfgang; Ross Joel S; Eggert Karla; Adler Georg  
 CS Philipps-University Marburg, Marburg, Germany.. oertelw@med.uni-marburg.de  
 SO Neurology, (2007 Jul 24) Vol. 69, No. 4 Suppl 1, pp. S4-9. Ref: 33  
 Journal code: 0401060. E-ISSN: 1526-632X.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 General Review; (REVIEW)  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 200708  
 ED Entered STN: 31 Jul 2007  
 Last Updated on STN: 17 Aug 2007  
 Entered Medline: 16 Aug 2007  
 AB Transdermal patches are used for the **treatment** of various  
 diseases including neurologic and psychiatric disorders such as Parkinson  
 disease (PD), major depression, and attention deficit hyperactivity  
 disorder. They are believed to offer many advantages over conventional  
 oral therapies. By providing smoother, continuous drug delivery and  
 steadier plasma levels, patches may reduce the incidence of side effects,  
 thus making optimal therapeutic doses easier to attain and potentially  
 improving **treatment** efficacy and compliance. Drug delivery  
 systems such as patches that are more patient- and caregiver-friendly may  
 enable patients to continue **treatment** for longer periods and to  
 attain greater, more sustained **treatment** benefits. To date,  
 approved therapies for **Alzheimer** disease (AD), including  
 cholinesterase inhibitors and memantine, are orally administered.  
 Potential advantages associated with patches provide a therapeutic  
 rationale to offer additional benefits in AD patients. Rivastigmine is  
 well suited to patch administration because it is a small, potent molecule  
 that is both lipophilic and hydrophilic. A rivastigmine patch has been  
 developed and may provide a promising new approach to dementia therapy.

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L16 ANSWER 6 OF 3143 MEDLINE on STN  
 AN 2007676993 IN-PROCESS  
 DN PubMed ID: 17997702  
 TI Immunotherapy as **treatment** for Alzheimer's disease.  
 AU Hawkes Cheryl A; McLaurin Joanne  
 CS Center for Research in Neurodegenerative Diseases, University of Toronto,  
 Toronto, Ontario, Canada.. cheryla.hawkes@utoronto.ca  
 SO Expert review of neurotherapeutics, (2007 Nov) Vol. 7, No. 11, pp.  
 1535-48. Ref: 152  
 Journal code: 101129944. E-ISSN: 1744-8360.  
 CY England: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 LA English  
 FS NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals  
 ED Entered STN: 20 Nov 2007  
 Last Updated on STN: 10 Dec 2007  
 AB Alzheimer's disease (AD) is a progressive neurodegenerative  
 disorder that is characterized pathologically by the deposition of  
 beta-amyloid (A beta)-containing extracellular neuritic plaques,  
 intracellular neurofibrillary tangles and neuronal loss. Much evidence  
 supports the hypothesis that A beta peptide aggregation contributes to AD  
 pathogenesis, however, currently approved therapeutic **treatments**  
 do nothing to stop or reverse A beta deposition. The success of active  
 and passive anti-A beta immunotherapies in both preventing and clearing  
 parenchymal amyloid in transgenic mouse models led to the initiation of an  
 active anti-A beta vaccination (AN1792) trial in human patients with  
 mild-to-moderate AD, but was prematurely halted when 6% of inoculated  
 patients developed aseptic meningoencephalitis. Autopsy results from the  
 brains of four individuals treated with AN1792 revealed decreased plaque  
 burden in select brain areas, as well as T-cell lymphocytes in three of  
 the patients. Furthermore, antibody responders showed some improvement in  
 memory task measures. These findings indicated that anti-A beta therapy  
 might still be a viable option for the **treatment** of AD, if  
 potentially harmful proinflammatory processes can be avoided. Over the  
 past 6 years, this target has led to the development of novel experimental  
 immunization strategies, including selective A beta epitope targeting,  
 antibody and adjuvant modifications, as well as alternative routes and  
 mechanisms of vaccine delivery, to generate anti-A beta antibodies that  
 selectively target and remove specific A beta species without evoking  
 autoimmunity. Results from the passive vaccination AD clinical trials  
 that are currently underway will provide invaluable information about both  
 the effectiveness of newly improved anti-A beta vaccines in clinical  
**treatment**, as well as the role of the A beta peptide in the  
 pathogenesis of the disease.

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